PAYING DOWN THE SLEEP DEBT: REALIZATION OF BENEFITS DURING SUBSEQUENT SLEEP RESTRICTION AND RECOVERY

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ABSTRACT

The study objective was to determine whether sleep extension (a) improves alertness and performance during subsequent sleep restriction and (b) mediates the rate at which alertness and performance are restored by post-restriction recovery sleep. Twenty-four healthy adult participants (ages 18-39) were randomly assigned to an Extended [10 hours time in bed (TIB)] or Habitual [mean (SD) = 7.09 (0.7)] sleep group for one week, followed by one Baseline (10 hours or habitual TIB), seven Sleep Restriction (3 hours TIB), and five Recovery Sleep nights (8 hours TIB) with performance [Psychomotor Vigilance Task (PVT)] and alertness [Maintenance of Wakefulness Test (MWT); Stanford Sleepiness Scale (SSS)] tests administered hourly throughout. We conclude that the extent to which sleep restriction impairs alertness and performance, and the rate at which these impairments are subsequently reversed by recovery sleep, varies as a function of the amount of nightly sleep obtained prior to the sleep restriction period.

1. INTRODUCTION

American adults report sleeping an average of 6.8 hours on weeknights (National Sleep Foundation, 2005) - considerably less than the 8 hours of sleep thought to be necessary to restore and sustain optimal daytime alertness. However, despite its near-ubiquitousness, chronic sleep restriction has not been scientifically studied to the same extent as acute, total sleep deprivation. In part, this has most likely been due to (a) the relative logistical difficulties associated with studying chronic sleep restriction in a controlled manner, and (b) an implicit, parsimonious assumption that the effects of chronic sleep restriction are qualitatively identical to those of acute total sleep deprivation, differing only in terms of the rate at which the deficits accrue.

In previous studies of chronic sleep restriction (7 or more consecutive nights) it has been shown that performance and alertness are degraded in a dose-dependent manner (Belenky et al., 2003; Van Dongen et al., 2003). Also apparent in both studies were (a) substantial individual differences in performance during (resilience to) sleep restriction, and (b) a failure for some aspects of performance to be restored to baseline levels after 3 nights of recovery sleep (with 8 hours time in bed per night). The latter finding was unexpected – extrapolating from total sleep deprivation studies (Lorenzo et al., 1995; Corsi-Cabrera et al., 1996; Rosa et al., 1983), it was anticipated that this amount of recovery sleep would produce greater levels of performance improvement than what was observed. Instead, this finding suggested the intriguing possibility that the neurobiological mechanism(s) underlying performance and alertness vary as a function of (and perhaps adapt to) habitual, nightly sleep duration, and that such changes have a relatively long time constant – e.g., requiring multiple (e.g., 7) days of continuously elevated sleep pressure (i.e., a longer duration than would typically be imposed in a formal total sleep deprivation study or be manifested in nature as a result of exposure to stressors). Consistent with the possibility of a slow-to-adapt physiological mechanism that mediates alertness and performance, cross-study comparisons of results from some other of our studies (Wesensten et al., 2005; Balkin et al, 2005) were also consistent with the possibility that recovery rate following multiple days of sleep restriction varies as a function of prior, habitual sleep duration.

Therefore, the present study was conducted to systematically determine the effects of prior sleep history on rates of performance and alertness degradation during chronic (7 nights) sleep restriction and during the subsequent recovery period. Specifically, it was predicted that "banking extra sleep" by extending nightly time in bed (TIB) would confer protective benefits during subsequent sleep restriction, and facilitate recovery from that sleep restriction.

2. METHODS

This study was approved by the Walter Reed Army Institute of Research Human Use Review Committee and the United States Army Medical Research and Materiel Command Human Subjects Research Review Board and was performed in accordance with the ethical standards of the 1964 Declaration of Helsinki.

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2.1 Participants

Civilian and active duty military men and women 18 to 39 years of age were recruited via flyers posted at local colleges and universities, and local military installations. After providing informed consent, participants completed questionnaires to determine eligibility based on physical state, psychological state, sleep habits, and chronotype. Participants also underwent a physical examination including a 12-lead electrocardiogram (ECG) and evaluation of blood and urine samples to determine general health, including pregnancy, and drug use. In order to reduce inter-subject variability in nighttime sleep, participants were excluded if they reported any of the following for the preceding month: (1) habitual daytime napping ($\geq$ one nap per week), (2) an average of more than seven hours sleep per night (the national average) Sunday through Thursday, (3) average nighttime lights-out times earlier than 2100 hours Sunday through Thursday, (4) average morning wake-up times later than 0900 Monday through Friday, or (5) time zone travel across more than three time zones within the last three months. Also, those with extreme morning (69) or extreme evening (31) chronotype scores (Horne and Ostberg, 1976) were excluded. To avoid withdrawal effects, participants who used nicotine regularly within the past three years or who consumed more than 400 mg caffeine daily (on average) were also excluded.

After eligibility to participate was ascertained, volunteers were randomly assigned to either the "Extended" or "Habitual" sleep group (described below) (n = 12 per group). Seven males and 5 females (mean age = 24.0 [stdev= 6.1]) were assigned to the Extended group; 4 males and 8 females (mean age = 26.0 [stdev = 7.1]) were assigned to the Habitual group. Power estimates calculated using Psychomotor Vigilance Task mean speed at baseline and sleep restriction Day 7 from a prior, similar sleep restriction study (Belenky et al., 2003) revealed that a sample size of 12 subjects per group would be sufficient to yield a power of 0.90.

2.2 Testing facilities

During testing and sleep periods, each subject was housed individually in a sound attenuated 8' x 10' room that included a bed and computer workstation. Ambient temperature was approximately 23 °C, and lighting was approximately 500 lux. Background white noise was 65 dB at all times. When not engaged in testing or sleep, participants remained in a common living area to play games, eat, read, or watch television and movies. Participants were monitored continuously by at least one laboratory technician.

2.3 Procedure

The study design consisted of 3 consecutive within-subjects' phases: (1) At-home, (2) In-laboratory Overnights, and (3) Full-Time In-laboratory. The phases outlined in Table 1 are described in more detail below.

Table 1. Study Design

PHASE	STUDY DAY	DAYS	HOURS IN BED	MEASURES
At-home	----	14	"Usual"	actigraphy, sleep diary, call-in
In-Lab OVERNIGHTS	O1-O7	7	Extended (10) or Habitual	actigraphy, sleep diary
Full-time In-Lab, BASELINE	B	1	Extended (10) or Habitual	actigraphy, PSG, PVT, MWT, SSS
Full-time In-Lab, RESTRICTION	SR1-SR7	7	3	actigraphy, PSG, PVT, MWT, SSS
Full-time In-Lab, RECOVERY	R1-R5	5	8	actigraphy, PSG, PVT, MWT, SSS

(1) *14-day At-home phase*. For 14 days prior to the in-laboratory phases, participants wore a wrist actigraph continuously, recorded their daily sleep times, and called into a time-stamped answering machine before and after nocturnal sleep periods. They were instructed to maintain their habitual sleep/wake schedule (usual nightly TIB); otherwise, volunteers were allowed to maintain their usual lifestyles.

(2) *7-day In-laboratory Overnight phase (O1—O7).* Immediately following the first phase, participants were randomly assigned to either a sleep "Extension" group (nightly TIB = 10 hours) or a "Habitual" sleep group (usual nightly TIB). "Habitual" sleep schedule was determined from actigraphy, sleep logs, and telephone call-ins the prior two weeks. During this phase, participants slept in the laboratory each night and both groups maintained a fixed wake time of 0700 for this and all subsequent phases. Sleep was recorded actigraphically. Participants left the laboratory during the day and maintained their usual day-time activities.

(3) *Full-Time In-Laboratory phase.* Following the seventh night of Extension or Habitual sleep described above, participants returned to the laboratory for the in-laboratory phase consisting of baseline, sleep restriction, and recovery sub-phases. Upon arrival at 1600 hours, they were briefed on study procedures, vital signs were taken and a urine sample collected for drug analyses in all participants and pregnancy screening in women. Polysomnographic (PSG) recording electrodes (electrooculogram [EOG], electromyogram [EMG], O_1, O_3, C_3, and C_4 electroencephalogram [EEG] sites) were applied and participants continued to wear wrist actigraphs. Participants also were given instructions and

practice on performance and alertness tasks described below. Upon awakening at 0700, vital signs were measured (and monitored during waking) and participants were allowed to eat a meal. Beginning at 0800, tests were administered every hour through 1800.

a. Baseline day (B). Timing of lights out was based on participants' average TIB the previous week with baseline testing the following day.

b. 7-day Sleep Restriction phase (SR1-SR7). Following the baseline testing day, participants began the 7-day Sleep Restriction phase in which nightly TIB was 0400-0700 hours followed by daytime testing from 0800 through 1800 hours.

c. 5-day Recovery phase (R1-R5). Following the last Sleep Restriction testing day, participants began the 5-day Recovery phase in which nightly TIB was 2300-0700 hours followed by daytime testing from 0800 through 1800 hours.

At the end of the fifth recovery day, vital signs were measured, all recording equipment removed, and a medical examination was performed. Participants were then debriefed and released from the study.

2.4 Measures

Actigraphy. Wrist movements were recorded using the Mini Motionlogger BMA-32 (Ambulatory Monitoring, Inc., Ardsley, NY). Data were scored for total sleep time (TST; minutes of sleep within the identified sleep period [elapsed time from the start of sleep to sleep end time]).

Polysomnography. Polysomnographic measurements included electroencephalogram [EEG (C3 AND C4)], electrooculogram [EOG (outer canthi of each eye)], and electromyogram [EMG (mental/submental)]. Contralateral mastoid leads served as references for all unipolar measurements (EEG and EOG). PSG data was scored in accordance with Rechtschaffen and Kales criteria (Rechtschaffen and Kales, 1968) using the Alice 4 Sleepware software (Respironics, Inc., Murraysville, PA). Dependent measures for nighttime sleep periods (defined as lights out to lights on) included minutes of individual stages [1, 2, slow wave sleep (SWS; stages 3 and 4 combined) and rapid eye movement sleep (REM)] and total sleep time [(TST) sum of minutes spent in all sleep stages]. The dependent measure for the Maintenance of Wakefulness Test (described next) was latency to the first 30-second epoch of stage 1 sleep.

Maintenance of Wakefulness Test (MWT). For the MWT, participants were escorted to their individual darkened, sound-attenuated bedrooms and allowed to lie down on their beds. They were instructed to close their eyes and to try to remain awake. PSG was monitored online. Participants were awakened at the onset of stage 2 sleep. If participants did not fall asleep after 20 minutes, the test was terminated.

PDA Psychomotor Vigilance Task (PVT). A 5-minute version of the Psychomotor Vigilance Task (PVT) (Thorne et al., 1985) was administered on a personal digital assistant (PDA). PVT was analyzed for speed (1/reaction time*1000), and number of lapses (reaction times $\geq$ 500 msec).

Stanford Sleepiness Scale (SSS). Participants selected which of seven statements best described their current state of alertness ranging from "1 – feeling active and vital; alert; wide awake" to "7 – almost in reverie; sleep onset soon; losing struggle to remain awake" (Hoddes et al., 1973). The dependent variable was self-rated sleepiness score.

2.5 Analyses

Nighttime Sleep. Nighttime sleep data (actigraphy and PSG) were analyzed using a mixed-model analysis of variance (ANOVA) in SPSS® Version 12.0 for PC (SAS Institute Inc., Cary NC). For nighttime actigraph sleep data, the model included fixed effects for Group (Extended v. Habitual) and Day (14 levels during the "at-home" sleep schedule assessment phase; 7 levels during the in-laboratory, overnight phase: O1-O7); for nighttime PSG sleep data, the model included fixed effects for Group and Day (13 levels across the Full-time, In-Laboratory sub-phases: B, SR1-SR7, R1-R5). Significant interactions were followed by post-hoc t-tests (Bonferroni correction). Greenhouse-Geisser corrections were applied to repeated measures effects. Statistical significance was $p < .05$.

Performance and Sleepiness. Responses at each time of day for PVT, MWT, and SSS variables were collapsed to obtain daily mean values. Time-of-day effects will be examined separately and presented elsewhere.

For PVT, MWT, and SSS variables, discontinuous growth modeling (DGM) (Singer and Willett, 2003) was used to examine patterns of responses across days of sleep restriction and recovery. DGM provides the ability to describe intra-individual patterns of change in terms of three distinct parameters—a sleep restriction slope (RESTRICT), a recovery transition parameter (TRANS), and a recovery slope (RECOV). Analyses were conducted using the open-source platform R (R Development Core Team, 2005) and the nonlinear and linear mixed effect model (NLME) package for R (Pinheiro and Bates, 2000). The growth modeling strategy used in the analyses is similar to that described

previously (Bliese and Ployhart, 2002; Bliese et al., 2006), and consists of several steps performed separately for each variable. During the first steps the nature of the intra-individual growth trajectories over time were identified and modeled, and the extent to which the growth trajectories contain reliable individual differences was determined. Identification of individual factors that explain individual differences in the intra-individual growth trajectories (i.e., group and age) were identified in subsequent steps.

In this model, the effects of experimental day were captured using three level 1 predictors. The first predictor (RESTRICT) was a vector of sequential whole numbers ranging from zero (baseline) to 12 (final recovery day). The second predictor (TRANS) was a dummy coded variable vector containing a value of zero for data collected during the sleep restriction phase, and a value of one for measures collected during the recovery phase. The third predictor (RECOV) was a vector containing zeros for measures collected during the sleep restriction phase and sequential numbers from zero to four for measures collected across the five recovery days. Group was included as a level 2 predictor of the level 1 predictors listed above. Effects of age were controlled for in the model.

3. RESULTS

Due to technical difficulties, one or more sessions of the various dependent measures were lost from some subjects from each sleep group. Because the analytical methods are robust to missing values, these subjects are included in the analyses but with consequently reduced degrees of freedom.

3.1 Nighttime Sleep

At-Home Phase. Actigraphically measured nightly total sleep time collapsed across all nights did not differ between Extended [mean (stdev) TST = 361 (113) min] and Habitual [mean (stdev) TST = 399 (86) min] groups during the initial two-week "At-home" phase ($P > 0.05$). Only TST on nights 6 and 8 differed between groups, with the Habitual group obtaining more sleep [Day 6: Extended mean (stdev) TST = 382 (29) and Habitual mean (stdev) TST = 469 (30); Day 8: Extended mean (stdev) TST = 329 (29) and Habitual mean (stdev) TST = 428 (30)].

In-Laboratory Overnight Phase. With the start of randomization to Extended v. Habitual groups, actigraphically recorded TST differed between groups [mean (stdev) TST minutes collapsed across nights = 479.5 (71.5) for Extended and 363.2 (63.9) for Habitual; group main effect $F_{1,21} = 49.12$, $p < 0.05$). The day and group x day effects were not significant ($P > 0.05$).

Full-time In-Laboratory Phase

Differences across days. Table 2 lists mean minutes of each PSG sleep variable as a function of day, in order from greatest to least, and collapsed across Group. In general, sleep amounts decreased from B to the sleep restriction phase, then increased from sleep restriction to recovery.

Table 2. Mean Sleep Stage Minutes And TST*

Var.														F-test
day														for Day
mins														
TST	B	R1	R2	R3	R4	R5	SR6	SR2	SR7	SR3	SR5	SR4	SR1	1432.5
	464	463	452	450	444	438	177	176	176	176	175	174	171	(12, 85)
Stg 1	B	R4	R5	R3	R2	R1	SR1	SR3	SR2	SR5	SR7	SR6	SR4	34.05
	44	34	33	31	27	20	9	6	6	6	6	5	5	(12, 55)
Stg 2	R1	B	R3	R2	R4	R5	SR1	SR7	SR2	SR5	SR3	SR6	SR4	287.1
	224	222	217	216	215	211	61	58	58	56	55	55	52	(12, 86)
SWS	R1	R2	R3	R5	R4	B	SR2	SR4	SR6	SR5	SR3	SR7	SR1	13.2
	122	106	99	99	97	91	83	83	82	82	82	81	72	(12, 112)
REM	B	R3	R2	R4	R5	R1	SR6	SR4	SR3	SR5	SR7	SR2	SR1	143.53
	107	104	104	101	98	96	35	35	34	33	32	30	30	(12, 156)

* Means that are not significantly different from each other are under the same bar.

Differences between Extended versus Habitual groups. At B, the Extended group obtained more TST (Day x Group, $F_{12,85} = 26.18$, $P < 0.001$; mean (SD), Extended = 520.5 (11.3), Habitual = 406.6 (12.1)], more REM (Day X Group, $F_{12,156} = 2.83$, $P = 0.002$), more stage 1 (Day x Group, $F_{12,55} = 5.79$, $P < 0.001$), and more stage 2 (Day x Group, $F_{12,86} = 6.50$, $P < 0.001$) than did the Habitual group.

No other effects were significant ($P > 0.05$).

3.2 Discontinuous growth modeling (PVT, MWT, SSS)

Step 1. The first step of the model captured the discontinuous, intra-individual component of the experimental design, followed by separate equations allowing for individual differences in responses across trials for the restriction, transition, and recovery parameters. An examination of the within-individual error structure was conducted (Bliese and Ployhart, 2002): These analyses suggested significant lag 1 serial autocorrelation in the repeated measures for lapses (-2 log-likelihood ratio = 21.75, $P < 0.0001$), speed (-2 log-likelihood ratio = 15.08, $P = 0.0004$), sleep onset latency (-2 log-likelihood ratio = 4.03, $P = 0.045$), and SSS score (-2 log-likelihood ratio = 6.28, $P = 0.01$); consequently a lag 1 within-individual error structure term was included for all models for each variable.

Step 2. The substantive variable of interest is the manipulation of prior sleep group; however, previous work has demonstrated the significant effect of age on responses

during sleep restriction (Bliese et al., 2006). Thus, age was included as a level two predictor of intercept and individual differences in the response slopes during the restriction, transition, and recovery phases in addition to sleep group. To test for variability in the three slope parameters, four models were contrasted for each variable: the first model restricted all three slope parameters to be equal across respondents, the second allowed individual slopes to vary for the restriction phase slope (RESTRICT), the third allowed for variability in both the restriction (RESTRICT) and the transition (TRANS) phases, and the fourth allowed for individual variability in all three parameters (RESTRICT, TRANS, RECOV). In all four models, age was included as an individual-level predictor of the intercept and slope parameters, so the subsequent tests reveal the extent to which residual variability is evident after the effects of age are controlled.

Table 3 provides degrees of freedom, model fit indices, log-likelihood ratios and P-values for the model contrasts for a) lapses, b) speed, c) sleep onset latency and d) SSS. For all variables, the best fitting model allowed for individual variability in the transition slope (TRANS) even after the effects of age were controlled. For speed, the fourth model could not be run because the residual variances were too small. Although residual individual differences in the restriction and recovery slopes were not significant for all variables based on the log-likelihood test, this test tends to be conservative[15] therefore the role of group (Extended versus Habitual) on all phases' slopes was examined.

Step 3. In the final step of modeling, the tests for variability in the three slope parameters were repeated as above with both group and age included as interaction terms for each variable.

For lapses, sleep onset latency, and SSS, allowing for variability in the TRANS parameter significantly improved the fit to the model (lapses, -2 log-likelihood ratio = 6.33, P = 0.04; sleep onset latency, -2 log-likelihood ratio = 8.05, P = 0.02; SSS, -2 log-likelihood ratio = 9.55, P = 0.01), so individual variability was allowed for the TRANS parameter in the final model for these three variables. For speed, allowing for variability in any parameter did not improve the fit to the model so individual variability was only allowed for the intercept in the final model.

3.3 Final model estimates

The final model estimates for each variable are provided in tables 4-5. The variance components provided in Tables 4-5 provide an estimate of the conditional intra-class correlation coefficient (ICC). As expected, age effects were significant and similar to effects reported by Bliese and colleagues (2006) (better performance in older subjects), and will not be discussed in detail here.

Psychomotor Vigilance Task . The model estimates for PVT (a) lapses and (b) speed, controlling for age, are displayed in Table 4. Figure 1 uses the parameter estimates from Table 4 to illustrate the experimental design effects for lapses and speed. The interactions between Group and each of three parameters (RESTRICT, TRANS, and RECOV) are illustrated in the figures. During sleep restriction, the Habitual group showed a steeper slope of PVT performance deterioration for lapses compared to the Extended group (RESTRICT x Group). At the transition from the sleep restriction to recovery phase, there were no differences between the groups for lapses, but for speed, the Extended group showed greater improvement compared to the Habitual group (TRANS x Group).

Table 3. Tests For Slope Variability In Design Effect Model

a) lapses

Model	*Random parameter*	*d.f.*	*AIC*	*log Lik*	*Test*	*L.ratio*	*P-value*
1	Intercept	12	1390.80	-683.40			
2	RESTRICT	14	1391.66	-681.83	1 vs. 2	3.13	0.21
3	RESTRICT, TRANS	17	1361.24	-663.62	2 vs. 3	36.43	0.00
4	RESTRICT, TRANS, RECOV	21	1355.82	-656.91	3 vs. 4	13.42	0.01

b) speed

Model	*Random parameter*	*d.f.*	*AIC*	*log Lik*	*Test*	*L.ratio*	*P-value*
1	Intercept	12	107.27	-41.63			
2	RESTRICT	14	105.89	-38.95	1 vs. 2	5.38	0.07
3	RESTRICT, TRANS	17	102.68	-34.34	2 vs. 3	9.21	0.03
4	RESTRICT, TRANS, RECOV	--	--	--	--	--	--

c) sleep onset latency

Model	*Random parameter*	*d.f.*	*AIC*	*log Lik*	*Test*	*L.ratio*	*P-value*
1	Intercept	12	1570.67	-773.33			
2	RESTRICT	14	1574.67	-773.33	1 vs. 2	0.0005	0.10
3	RESTRICT, TRANS	17	1565.67	-765.83	2 vs. 3	15.00	0.002
4	RESTRICT, TRANS, RECOV	21	1567.05	-762.53	3 vs. 4	6.62	0.16

d) SSS

Model	*Random parameter*	*d.f.*	*AIC*	*log Lik*	*Test*	*L.ratio*	*P-value*
1	Intercept	12	718.22	-347.11			
2	RESTRICT	14	719.43	-345.72	1 vs. 2	2.79	0.25
3	RESTRICT, TRANS	17	715.45	-340.72	2 vs. 3	9.96	0.02
4	RESTRICT, TRANS, RECOV	21	719.74	-338.87	3 vs. 4	3.71	0.45

During the 5-day Recovery phase the groups showed significantly different patterns of performance for lapses (RECOV x Group): the Extended group recovered significantly after one night and maintained a stable level of improved performance while the Habitual group gradually improved across the 5 Recovery days. While not significant, the pattern for PVT speed was like that of lapses.

Table 4. PVT Model Estimates

a) lapses	*Parameter*	*SE*	*d.f.*	*t-value*	*P-value*
Fixed effects					
Intercept (ms)	0.23	3.35	273	0.07	0.47
Restriction slope (RESTRICT)*	2.70	0.40	273	6.80	0.00
Age	-0.05	0.11	21	-0.40	0.35
Transition to recovery (TRANS)*	-15.33	2.70	273	-5.67	0.00
Recovery slope (RECOV)*	-3.14	0.88	273	-3.54	0.00
Prior sleep group (Group)	1.03	1.43	21	0.72	0.24
RESTRICT X Age*	-0.09	0.01	273	-6.75	0.00
Age X TRANS*	0.44	0.09	273	4.86	0.00
Age X RECOV*	0.12	0.03	273	4.02	0.00
RESTRICT X Group*	0.40	0.17	273	2.35	0.01
TRANS X Group	-0.85	1.16	273	-0.73	0.23
RECOV X Group*	-0.75	0.39	273	-1.94	0.03
	Correlations				
Variance components					
Intercept	8.70				
Transition to recovery	2.45	-0.66			
Residual	4.75				
Fit indices					
Deviance (-2 Log-lik)	-677.96				
AIC	1389.91				
BIC	1452.53				

b) speed	*Parameter*	*SE*	*d.f.*	*t-value*	*P-value*
Fixed effects					
Intercept (ms)*	3.68	0.53	273	6.88	0.00
Restriction slope (RESTRICT)*	-0.34	0.05	273	-7.34	0.00
Age	0.02	0.02	21	1.03	0.16
Transition to recovery (TRANS)*	1.72	0.26	273	6.66	0.00
Recovery slope (RECOV)*	0.33	0.10	273	3.22	0.00
Prior sleep group (Group)	-0.20	0.23	21	-0.89	0.19
RESTRICT X Age*	0.01	0.00	273	4.81	0.00
Age X TRANS*	-0.03	0.01	273	-3.22	0.00
Age X RECOV*	-0.01	0.00	273	-2.71	0.00
RESTRICT X Group	0.01	0.02	273	0.64	0.26
TRANS X Group*	-0.23	0.11	273	-2.10	0.02
RECOV X Group	0.05	0.05	273	1.12	0.13
Variance components					
Intercept	0.26				
Residual	0.06				
Fit indices					
Deviance (-2 Log-lik)	-45.90				
AIC	121.81				
BIC	177.06				

*p < .05, one-tailed

Maintenance of Wakefulness Test. The model estimate for MWT sleep latency, controlling for age, is displayed in Table 5. Figure 2 uses the parameter estimates from Table 5 to illustrate the experimental design effects for sleep latency. The significant interaction between Group and RESTRICT is illustrated in the figure. During sleep restriction, the Habitual group showed shorter sleep latency compared to the Extended group (RESTRICT x Group). The groups did not differ at the transition from the sleep restriction to recovery phase, nor in patterns of sleep latency during Recovery (TRANS x Group n.s.; RECOV x Group n.s.).

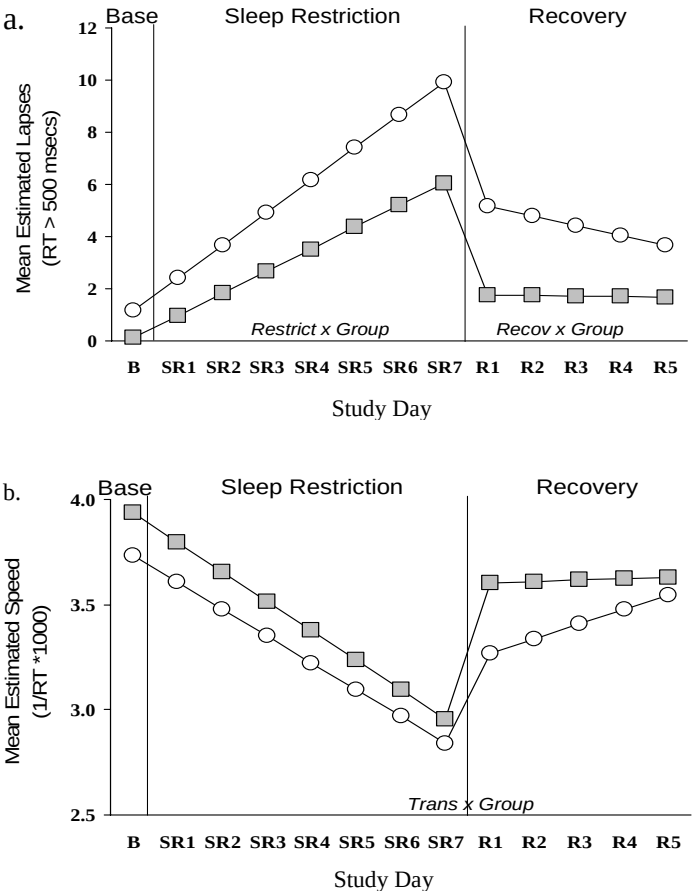

Fig. 1. Predicted psychomotor vigilance test a) lapses and b) speed for Extended (shaded squares) and Habitual (open circles) sleep groups controlling for age.

Stanford Sleepiness Scale. The model estimates for SSS self-rated sleepiness score, controlling for age, are displayed in Table 6. There were no significant interactions between Group and any of the three parameters (RESTRICT, TRANS, and RECOV).

4. DISCUSSION

One week of sleep extension improved resilience on measures of performance and alertness during

subsequent sleep restriction, and facilitated recovery thereafter. Discontinuous growth modeling was used to compare and contrast patterns of performance degradation across 7 days of sleep restriction (3 hrs TIB) for two groups of participants – one in which habitual sleep duration had been maintained for the previous 7 days (Habitual group), and the other in which TIB had been increased to 10 hrs for the previous 7 days (Extended group).

Table 5. Model Estimates For MWT Sleep Latency

	Parameter	*SE*	*d.f.*	*t-value*	*P-value*
Fixed effects					
Intercept (ms)	4.35	2.63	276	1.65	0.05
Restriction slope (RESTRICT)	-0.91	0.48	276	-1.90	0.03
Age*	0.38	0.09	21	4.31	0.00
Transition to recovery (TRANS)	0.66	3.25	276	0.20	0.42
Recovery slope (RECOV)*	3.11	1.07	276	2.90	0.00
Prior sleep group (Group)*	-3.56	1.13	21	-3.15	0.00
RESTRICT X Age*	-0.05	0.02	276	-3.05	0.00
Age X TRANS*	0.34	0.11	276	3.10	0.00
Age X RECOV	-0.00	0.04	276	-0.03	0.49
RESTRICT X Group*	0.68	0.20	276	3.32	0.00
TRANS X Group	-1.83	1.39	276	-1.31	0.10
RECOV X Group	-0.48	0.46	276	-1.04	0.15
	Correlations				
Variance components					
Intercept	2.90				
Transition to recovery	1.73	0.99			
Residual	7.53				
Fit indices					
Deviance (-2 Log-likelihoo	-763.20				
AIC	1560.40				
BIC	1623.20				

*p < .05, one-tailed

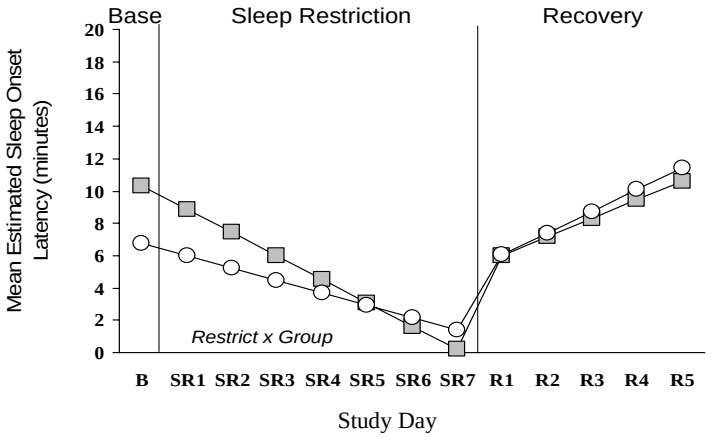

Fig. 2. Predicted sleepiness scores for MWT sleep latency for Extended (shaded squares) and Habitual (open circles) sleep groups controlling for age.

This approach allowed: (a) examination of the trajectories (slope) of performance degradation over the sleep restriction period, and the performance improvement associated with the transition and recovery periods. It also allowed us to determine the extent to which prior sleep duration is associated with systematic differences in performance and alertness trajectories. Differences between the Extended and Habitual groups for all three parameters (restriction slope, transition, and recovery slope) were evident.

Table 6. Model Estimates For SSS Scores

	Parameter	*SE*	*d.f.*	*t-value*	*P-value*
Fixed effects					
Intercept (ms)*	2.06	0.89	276	2.32	0.01
Restriction slope (RESTRICT)	0.15	0.11	276	1.30	0.10
Age	-0.02	0.03	21	-0.57	0.29
Transition to recovery (TRANS)	-0.51	0.89	276	-0.57	0.29
Recovery slope (RECOV)	-0.09	0.26	276	-0.35	0.36
Prior sleep group (Group)	0.34	0.38	21	0.88	0.20
RESTRICT X Age	0.00	0.00	276	1.00	0.16
Age X TRANS	-0.04	0.03	276	-1.17	0.12
Age X RECOV	-0.00	0.01	276	-0.14	0.45
RESTRICT X Group	-0.02	0.05	276	-0.37	0.36
TRANS X Group	-0.35	0.38	276	-0.91	0.18
RECOV X Group	-0.09	0.11	276	-0.85	0.20
	Correlations				
Variance components					
Intercept	0.59				
Transition to recovery	0.32	-0.77			
Residual	0.42				
Fit indices					
Deviance (-2 Log-likelihoo	-342.50				
AIC	719.00				
BIC	781.79				

*p < .05, one-tailed

Because our (and others') previous work shows that age accounts for a significant portion of the variance in performance during sleep loss (Bliese et al., 2006) this factor was controlled in the present study. As found previously, younger individuals in the current study showed a greater decline in PVT performance than did older individuals. Similarly, younger individuals also showed a greater increase in physiological (objectively measured) sleepiness. Age was not, however, a significant predictor of subjective sleepiness.

Differences in the polysomnographically measured sleep of the Habitual and Extended groups were evident on the baseline night on all sleep measures except SWS amount. Specifically, the Extended group had greater amounts of TST, REM, NREM, stage 1, and stage 2, compared to the Habitual group (as expected, given the longer TIB of this group) on this night. No group differences in sleep architecture were found during the restriction and recovery phases. Actigraphy data from the at-home period showed that the groups did not significantly differ prior to the random assignment to groups.

The present results suggest that apparent differences in the rates of recovery of alertness/performance following sleep restriction in previous studies (Belenky et al., 2003; Wesensten et al., 2005; Balkin et al., 2005) may have been due to differences in the amount of nightly sleep habitually obtained prior to the sleep restriction period (in one study, nightly pre-restriction sleep was extended, and in the other it was not). In the present study, performance deficits (PVT lapses and speed) recovered after one night of recovery sleep in the Extended Group. It should be noted that response speed failed to recover to "baseline" level for the Extended group. This is most likely because the "baseline" measures were in this case obtained following a week of extended sleep, whereas the recovery consisted of 8 hours TIB per night – i.e., allowing a more typical amount of nighttime sleep and resulting in a more typical level of performance. Accordingly, one night of recovery sleep in the Extended group restored mean response speed to a stable level that was comparable to that exhibited by the Habitual group at baseline. In contrast, the Habitual group showed continuing improvement (i.e., reductions in PVT lapses) across the five recovery days, and performance in this group failed to improve to same extent as that of the Extended group, even after 5 nights of recovery sleep.

In many previous sleep loss studies, one or two nights of sleep extension/adaptation are administered prior to the sleep loss phase. Findings from the present study suggest that this may not be adequate – i.e., that the long-term, habitual sleep duration of study participants can mediate their sensitivity/resiliency during sleep loss and subsequent recovery, so this factor should always be controlled or taken into account when studies are performed for the purpose of documenting and (especially) quantifying the effects of sleep loss on various aspects of alertness and performance.

Of course, findings from the present study also have implications for (a) mathematical modeling efforts to predict the effects of sleep/wake schedules on performance in operational settings, and (b) our understanding of the nature of the physiological processes that underlie alertness and performance. For example, the finding that prior nightly sleep duration impacts performance and sleepiness during subsequent sleep restriction and recovery is consistent with the assertions of Johnson and colleagues (2004) that a simple sleep reservoir conception – in which alertness and performance vary simply as a function of the extent to which an individual's idiosyncratic and consistent need for sleep has been satisfied (combined with the circadian rhythm of alertness) – is not adequate for describing and predicting alertness and performance during sleep restriction and recovery. Indeed, the present findings are consistent with their assertion that the homeostatic process modulating sleep need varies over time, albeit with a long time constant.

While recovery to sleep restriction is generally slow compared to total sleep loss, there is, of course, variability in both recovery rate and response to sleep restriction among individuals. Van Dongen and colleagues have shown that 'vulnerability' to the effects of total sleep loss is a trait-like characteristic and that individuals show stability in their response with repeated testing (Van Dongen et al., 2004). In the present study, the demonstrated greater "sensitivity" of the Habitual versus Extended sleep group suggests the possibility that some of the observed interindividual difference may not be "sensitivity" or "vulnerability" per se, but rather habitual sleep duration. That is, habitually shorter sleepers may demonstrate increased sensitivity to sleep loss when faced with a challenge (i.e., period of sleep loss) – a vulnerability that is based as much or more on their habitual sleep behavior than on differences in their inherent vulnerability to the effects of sleep loss. In other words, the "trait" may be, at least in part, how much sleep is typically obtained rather than (or in addition to) how much sleep loss can be effectively tolerated. Consistent with this possibility, Klerman and Dijk (2005) have shown that habitually shorter sleepers fall asleep faster on MSLTs and obtain more 'recovery' sleep in a sleep extension protocol.

Similarly, recent evidence suggests that individuals with a PER3 clock gene polymorphism are more susceptible to sleep-loss induced performance impairments (Viola et al., 2007). Like habitually short sleepers, the sleep of these susceptible individuals is also characterized by high initial values of SWA and of theta during wakefulness. Taken together, these studies suggest an overlap between habitual prior sleep duration and trait-sensitivity to sleep loss. For example, it is possible that the PER3 polymorphism actually mediates the sleep homeostat indirectly (e.g., via the timing of sleep periods and/or the level of sleep debt carried), and that it is this "behavioral" effect of the PER3 polymorphism that ultimately determines an individual's sensitivity/resilience to the effects of sleep loss.

One important, and perhaps critical, aspect of the present study that should be taken into consideration is the fact that "recovery sleep" was restricted to 8 hours TIB. It is likely that recovery would have been faster in both groups had they been afforded a longer nightly recovery sleep opportunity. Future studies varying both duration of recovery and degree of sleep restriction (e.g., 5 instead of 3 hours TIB) will be needed to fully delineate the effects of prior sleep extension, and to accurately determine the amount of recovery sleep needed following extended periods of sleep restriction.

In summary, the present study demonstrates beneficial effects of prior sleep extension on performance and alertness during sleep restriction and during subsequent recovery from that sleep restriction. One implication of this study is that habitual sleep duration needs to be taken into consideration when determining individual differences in susceptibility to sleep loss. From a practical standpoint, the present findings suggest that the "banking" of sleep prior to sleep loss may help sustain performance and alertness in operational environments and speed recovery (i.e., improve "recycle rate" of operators).

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